

## Complete Summary

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### GUIDELINE TITLE

Coronary heart disease (CHD): symptoms, diagnosis and treatment.

### BIBLIOGRAPHIC SOURCE(S)

Finnish Medical Society Duodecim. Coronary heart disease (CHD): symptoms, diagnosis and treatment. In: EBM Guidelines. Evidence-Based Medicine [CD-ROM]. Helsinki, Finland: Duodecim Medical Publications Ltd.; 2006 Apr 28 [Various].

### GUIDELINE STATUS

This is the current release of the guideline.

This guideline updates a previous version: Finnish Medical Society Duodecim. Coronary heart disease (CHD): symptoms, diagnosis and treatment. In: EBM Guidelines. Evidence-Based Medicine [CD-ROM]. Helsinki, Finland: Duodecim Medical Publications Ltd.; 2006 Mar 14 [Various].

## COMPLETE SUMMARY CONTENT

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## SCOPE

### DISEASE/CONDITION(S)

- Stable angina pectoris
- Coronary heart disease (CHD)

### GUIDELINE CATEGORY

Diagnosis  
 Evaluation  
 Prevention

Risk Assessment  
Treatment

#### CLINICAL SPECIALTY

Cardiology  
Family Practice  
Internal Medicine  
Pharmacology

#### INTENDED USERS

Health Care Providers  
Physicians

#### GUIDELINE OBJECTIVE(S)

Evidence-Based Medicine Guidelines collect, summarize, and update the core clinical knowledge essential in general practice. The guidelines also describe the scientific evidence underlying the given recommendations.

#### TARGET POPULATION

Individuals with suspected or confirmed stable angina and coronary heart disease

#### INTERVENTIONS AND PRACTICES CONSIDERED

##### Diagnosis

1. Assessment of symptoms of angina (pectoris) including classification of chest pain
2. Physical investigations (blood pressure, heart rate, heart sounds, palpation, presence of pallor, signs of heart failure, other physical signs)
3. Electrocardiography
4. Laboratory investigations (serum lipids, blood glucose, blood haemoglobin, chest x-ray, myocardial enzymes or markers)
5. Exercise tolerance test
6. Radionuclide imaging
7. Exercise echocardiography
8. Coronary angiography
9. Special diagnostic considerations in women

##### Assessment/Treatment of Risk Factors

1. Assessment of modifiable and nonmodifiable risk factors
2. Drugs (aspirin, a beta-blocker, a statin) to slow down atherosclerosis and prevent myocardial infarction
3. Folic acid (and vitamins B6 & B12) to lower elevated homocysteine levels (a risk factor for accelerated atherogenesis)
4. Smoking cessation
5. Treatment of hypertension

6. Assessment of lipid profile and reduction of hyperlipidaemia by using statins
7. Treatment of obesity, including recognition of metabolic syndrome and consideration of combination therapy with a statin and fibrate
8. Physical exercise
9. Comprehensive cardiac rehabilitation including combination of exercise, psychological and educational interventions

## Treatment

1. Aspirin (clopidogrel for patients not responding to aspirin)
2. Low-molecular-weight heparin
3. Coronary care unit monitoring
4. Sublingual or aerosol nitrates
5. Long-acting nitrates (a nitrate patch)
6. Beta-blockers, such as carvedilol
7. Angiotensin-converting enzyme (ACE) inhibitor in combination with beta-blocker
8. Dihydropyridine derivatives (amlodipine, felodipine, isradipine) in combination with beta-blockers
9. Calcium antagonists, such as diltiazem, nifedipine, lidoflazine, and verapamil
10. Revascularization (percutaneous transluminal coronary angioplasty [PTCA], coronary artery bypass grafting [CABG] surgery, off-pump coronary artery bypass [OP-CAB] grafting, coronary stenting, corticosteroids for postpericardiotomy syndrome [PPS])

## MAJOR OUTCOMES CONSIDERED

- Sensitivity and specificity of diagnostic tests
- Risk reduction for cardiovascular events and death
- Mortality (total or overall)
- Coronary heart disease mortality
- Morbidity
- Reductions in blood pressure
- Reductions in smoking
- Reductions in blood lipids
- Improvements in exercise tolerance
- Incidence of cardiac events
- Incidence of reinfarction
- Restenosis rate
- Arterial graft patency
- Adverse effects
- Cardiac death rate
- Number of angina episodes per week

## METHODOLOGY

### METHODS USED TO COLLECT/SELECT EVIDENCE

Hand-searches of Published Literature (Primary Sources)  
 Hand-searches of Published Literature (Secondary Sources)  
 Searches of Electronic Databases

## DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

The evidence reviewed was collected from the Cochrane database of systematic reviews and the Database of Abstracts of Reviews of Effectiveness (DARE). In addition, the Cochrane Library and medical journals were searched specifically for original publications.

## NUMBER OF SOURCE DOCUMENTS

Not stated

## METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Weighting According to a Rating Scheme (Scheme Given)

## RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Levels of Evidence

- A. Strong research-based evidence. Multiple relevant, high-quality scientific studies with homogenic results.
- B. Moderate research-based evidence. At least one relevant, high-quality study or multiple adequate studies.
- C. Limited research-based evidence. At least one adequate scientific study.
- D. No research-based evidence. Expert panel evaluation of other information.

## METHODS USED TO ANALYZE THE EVIDENCE

Review of Published Meta-Analyses  
Systematic Review

## DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Not stated

## METHODS USED TO FORMULATE THE RECOMMENDATIONS

Not stated

## RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Not applicable

## COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

## METHOD OF GUIDELINE VALIDATION

Peer Review

## DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

Not stated

## RECOMMENDATIONS

### MAJOR RECOMMENDATIONS

The levels of evidence [A-D] supporting the recommendations are defined at the end of the "Major Recommendations" field.

#### Basic Rules

- A history, clinical examination, and some basic tests are usually sufficient for clinical diagnosis of stable angina (pectoris).
- The diagnosis is often confirmed when prescribed drug therapies alleviate symptoms.
- When the diagnosis is not clear, an exercise tolerance test (ETT) is required.
- An ETT can be used to assess the severity of the disease and the working capacity of the patient.
- An ETT is also required when revascularization (Yusuf et al., 1994; Sudlow et al., 2002; Davies, et al., 1997) [A] is considered.
- Severe, newly-diagnosed or progressive (unstable) angina often requires urgent hospitalisation and sometimes angioplasty revascularisation.
- The treatment of coronary heart disease (CHD) includes the evaluation of the overall risk. Management of risk factors is causal treatment.
- The basic medication for most patients consists of nitrates, aspirin, beta-blocker, an angiotensin-converting enzyme (ACE) inhibitor, and a statin.

#### Epidemiology

- Under the age of 65, CHD mortality in men is three times that of women. In the older age group, the mortality of both genders is equal. After the age of 80 the CHD mortality of women is twice that of men.
- Total CHD mortality remains unchanged but mortality under the age of 65 has fallen by 50% over the past two decades (Finland).

#### Clinical Manifestations of CHD

- Chest pain is the most common presentation.
- Other disease manifestations are dyspnoea or collapse on exertion, arrhythmias, unstable angina, acute or chronic heart failure, myocardial infarction, and sudden death.

#### Symptoms and Clinical Diagnosis

- For differential diagnosis of chest pain, see the Finnish Medical Society Duodecim guideline "Differential Diagnosis of Chest Pain."

- Stable angina is a clinical diagnosis which indicates the repeated occurrence of chest pain, induced by an exercise level typical for the patient. The pain is relieved by rest and shows no great daily variation; variation is typical for nonischaemic chest pain.
- Typical angina pain
  - Is precipitated on exertion
  - Becomes worse as the exertion continues
  - Is felt across a wide area in the middle of the chest (not in the cardiac apex) and is tight and constrictive in nature
  - May be transmitted to the neck, jaw, arms, epigastric region, or back
  - May become worse in cold weather, after a heavy meal, or during static work
  - Is relieved in a few minutes by rest or glyceryl trinitrate
- However, only about half of all patients have a typical presentation of the symptoms.
- The patient's history of pain is more reliable for diagnosis in men than in women aged less than 50 years. The probability of CHD in males over 55 with typical symptoms is 90%.
- Classification of chest pain (Canadian Cardiovascular Society [CCS] -- corresponds to the previously used New York Heart Association [NYHA] classification) (see Table 1 in the original guideline document)
- Dyspnoea on exertion may be the presenting symptom instead of chest pain. Dyspnoea is caused by temporary heart failure brought on by the exertion.
- Ischaemic pain or dyspnoea forces the patient to slow down or stop walking.
- Nocturnal chest pain is, in most cases, a symptom of gastrooesophageal reflux.
- Some patients describe the pain as burning, which might be misinterpreted as oesophageal pain.
- The pain is usually induced by predictable levels of exercise when the rate-pressure product exceeds the patient's individual threshold. However, in some patients exercise tolerance may vary to some degree, but in general, totally asymptomatic days are rare. Large variations are typical of nonischaemic pain.
- The pain may also be precipitated by mental stress, as this causes rate-pressure product increases.
- The pain may be triggered by rapid onset of walking. After warming up, the patient can again "walk through his angina."
- The transmission of pain does not vary.
- After more intense exercise the pain may remain for 15 minutes. More prolonged pain should be considered as myocardial infarction or delayed recovery from an ischaemic insult (stunning).
- The above text refers to stable angina, for unstable angina see the Finnish Medical Society Duodecim guideline "Unstable Angina Pectoris."

#### Atypical Chest Pain Not Suggestive of Coronary Heart Disease

- Appears also at rest
- Exercise tolerance is good despite pain
- Continues for hours or days
- Is associated with breathing or chest wall movements
- Is sharp in character
- Is displaced laterally towards the apex

- May be felt on palpation
- Is experienced as palpitations or occasional ectopic beats
- Is felt in the upper abdominal region or below the left costal arch
- Is not relieved with glyceryl trinitrate within a few minutes

## Investigations

### Physical Examination

- In most patients, physical examination is totally normal.
- Check blood pressure and heart rate (might be elevated due to pain).
- Listen for systolic bruit: CHD patients often have concomitant carotid disease or generalised arteriosclerotic disease.
- Aortic stenosis is frequently associated with CHD.
- S3 and a soft mitral regurgitant murmur are signs of impaired cardiac function. They may also be a transient functional effect of prolonged ischaemia or may only be audible during exercise.
- Cardiac palpation may reveal left ventricular hypertrophy (LVH). A patient with LVH will experience angina even in mild coronary heart disease.
- Pallor may suggest anaemia.
- Transient signs of heart failure may appear after a prolonged ischaemic attack.

### Electrocardiogram (ECG)

- The ECG is normal at rest in 30 to 50% of the patients.
- ST-T changes are a sensitive finding but nonspecific.
- Slightly prolonged (<0.24 sec) PR interval is common.
- Patients with LVH often suffer from ischaemia and angina.
- Left bundle branch block (LBBB) is suggestive of CHD or hypertrophy, or both.
- A previous infarction is a definite sign of CHD.
- A reversible ST segment depression, which appears during pain, is strong evidence for CHD.
- Continuous monitoring in the coronary care unit (CCU) or by the Holter method may reveal silent ischaemia (ST depression). Silent ischaemia is more common than symptomatic ischaemia but it is not harmless, and its diagnosis is dependent on the Holter technique (Tresch, 1995) [B]. The assessment of silent ischaemia with Holter monitoring is difficult and technically demanding. In the diagnosis of ischaemia its significance is limited to risk stratification of a patient with unstable angina.

### Laboratory Investigations

- Risk factor investigations: Serum lipids (Institute for Clinical Systems Improvement [ICSI], 2003; Health Technology Assessment [HTA]-20030537, 2004) [A], and blood glucose
- Haemoglobin
- Chest x-ray: heart failure, valvular calcification, and other causes of chest pain
- A small rise in cardiac enzymes or markers is possible after a prolonged angina attack, even in the absence of actual infarction. Such a rise is often a

serious sign and is predictive of future infarction. Further investigations are necessary (see the Finnish Medical Society Duodecim guideline "Unstable Angina Pectoris").

#### Exercise Stress Test

- Exercise stress test or exercise tolerance test (ETT) is the most common test carried out in angina patients. However, it does have limitations and interpretation problems.
- ETT will assist in determining the severity of the disease (see Table 1 in the original guideline document).

#### Radionuclide Imaging

- The sensitivity is higher than, but the specificity is equal to, that of the ETT.
- Should be considered when ETT is normal but the disease is highly likely
- Valuable for patients with mobility problems
- First-line choice when an abnormal ECG impairs the interpretation of ETT

#### Exercise Echocardiography

- Ischaemia induces myocardial wall motion abnormalities, which can be detected with sensitive imaging equipment. When carried out by an experienced operator, the test is considered to be even more sensitive and accurate than ETT. Particularly good around the anterior wall area
- Useful when ECG is non-diagnostic because of abnormalities

#### Coronary Angiography

- The gold standard for preoperative investigation and CHD diagnostics. Nowadays used increasingly for diagnostic purposes.

#### Special Diagnostic Problems in Women

- The diagnostic specificity of chest pain in premenopausal women is low, unless the pain is clearly typical angina pain.
  - In typical angina, the pain or dyspnoea appear only on exertion.
  - Only about half of the women with typical angina pain have a significant CHD.
  - Premenopausal women complain of chest pain more often than men. The pain is usually atypical, i.e., appears in rest or is multiform.
  - In old age, the diagnostic sensitivity of the symptoms becomes as high as in males (90%).
- The predictive value of ETT is worse in women because of ST change due to sympathicotonic response. The number of false-positive test results is high in premenopausal women.
  - Performing an ETT should be avoided if there are no risk factors or typical angina pain.
  - An ST change resembling ischaemia with normal angiography finding (syndrome-X) is much more common in premenopausal women than in men.



- If the ischaemic ST-segment depression persists several minutes after the end of exertion, the positive predictive value of the test is good. If there is no ST segment change, the negative predictive value of the test is good.
- Under the age of 50, the specificity of radionuclide imaging may be higher than that of the conventional ETT.

### Treatment of CHD Risk Factors

- It is difficult to obtain firm evidence, based on extensive follow-up studies, on the benefits of modifying a single risk factor. The conclusions are based on epidemiological observations and pathophysiology (Pyörälä, 1996).
- To improve prognosis, minimizing all risk factors to slow down atherosclerosis and prevent myocardial infarction (MI) is valuable. Efficient secondary prevention usually includes aspirin, a beta-blocker, a statin, and discontinuation of smoking (Pyörälä, 1996; "MRC/BHF Heart Protection Study," 2002; Pitt et al., 1999).
- Smoking should be stopped (Critchley & Capewell, 2003) [A]. The risk of MI is 3-fold in smokers and even higher in women (Ounpuu, Negassa, & Yusuf, 2001). Alcohol consumption should be limited to moderate amounts.
- Hypertension should be treated. The target level of below 140/90 mmHg should be achieved. According to the latest study, the optimal level is 138/83 mm HG. Further is neither more beneficial (except in diabetics) nor harmful (Hansson et al., 1998).
- Effective reduction of hyperlipidaemia often requires the prescription of a statin. Target levels below are only meant as a guideline. According to the Heart Protection Study (HPS) in particular, hyperlipidaemia should be treated in high-risk patients, who will benefit from a statin even when their initial cholesterol level is nearly normal ("MRC/BHF Heart Protection Study," 2002). Statins have an effect on endothelial dysfunction of coronary arteries (Pitt et al., 1999), as well as on the inflammatory reaction and thrombosis ("Randomised trial of cholesterol lowering," 1994).
- Recommendations:
  - Serum cholesterol concentration below 5.0 mmol/L
  - Low-density lipoprotein (LDL) concentration below 3.0 mmol/L. Optimal level below 2.5 mmol/L.
  - Serum triglyceride concentration below 2 mmol/L
  - Serum cholesterol/serum high-density lipoprotein (HDL) below 4 mmol/L. Serum HDL in men >0.9 mmol/L and in women >1.1 mmol/L
  - See the related Finnish Medical Society Duodecim guideline on "Drug Treatment for Hyperlipidaemias" for details on relevant drug therapy.
- Treating obesity
  - Weight should be reduced to achieve a body mass index (BMI) <28. Patients with abdominal obesity (high waist-to-hip ratio) are particularly at a high risk (Yusuf et al., 2005) [B].
  - Recognise metabolic syndrome and consider starting combination therapy with a statin and fibrate. Such treatment warrants the monitoring of liver enzyme and creatine kinase (CK) values.
- Physical exercise
  - Regular exercise improves the sense of well being and prognosis by reducing many risk factors (U.S. Department of Health and Human Services [DHHS], 1995; Kugler, Seelbach, & Kruskemper, 1994;

Scottish Health Purchasing Information Centre, 1998; NHS Centre for Reviews, 1998; ICSI, 2002.; HTA-989125, 2004; HTA-998327, 2004; HTA-20030540, 2004) [A] (see the Finnish Medical Society Duodecim guideline "Physical Activity in the Prevention, Treatment and Rehabilitation of Diseases"). Physical activity also plays a part in primary prevention (Murphy et al., 2002) [C].

- Intense physical strain should be avoided.
- Based on a randomised secondary prevention study (Heart and Estrogen/Progestin Replacement Study [HERS]) and a primary prevention study (Women's Health Initiative [WHI]) (Rossouw et al., 2002) [A], hormone replacement therapy (HRT) offers no benefit.
- Antioxidant therapy with vitamin E had no beneficial effect in the HPS study ("MRC/BHF Heart Protection Study," 2002) and neither did vitamins A and C.
- Elevated serum homocysteine concentration is associated with vascular diseases; however, it does not appear to act as a predictor of arterial disease in healthy individuals (Knekt et al., 2001; ICSI, 2003; HTA-20030537, 2004) [C]. Nevertheless, homocysteine concentration correlates positively with blood pressure, cholesterol concentration, and smoking and is thus an indicator of the severity of the atherosclerotic process. The correlation has been explained by atherosclerosis-induced renal insufficiency leading to reduced homocysteine clearance, which, in turn, will increase its plasma concentration. Folic acid (and vitamins B6 and B12) lower serum homocysteine concentration, but evidence on its effect in slowing down the progression of vascular disease is scant (only one study in which the administration of vitamins after percutaneous transluminal coronary angioplasty [PTCA] lowered the incidence of restenosis) (Schnyder, et al., 2002) [B]. Several studies on secondary prevention are ongoing, but so far there is no evidence that vitamin substitution would reduce the incidence of cardiovascular diseases (Hung et al., 2003).
- Age, male gender, and family history of CHD are nonmodifiable risk factors. They must be included in the stratification of overall risk.
  - Female risk factors for CHD comprise diabetes, smoking, low HDL and genotype.

#### Pharmacotherapy: Modes of Action and Aims

- Myocardial ischaemia is reduced by lowering blood pressure and heart rate. Beta-blockade is sufficient when heart rate is down to 60 to 50 beats per minute (bpm). The treatment of hypertension aims at an optimal pressure, which according to the Hypertension Optimal Treatment (HOT) study (Hansson et al., 1998) is 138/83 mmHg.
- Aspirin is not prescribed for symptom relief. It reduces the blocking of coronary arteries. Aspirin is recommended for all patients with CHD at the dose of 75 to 150 mg/day, unless it is contraindicated (Anti-thrombotic Trialists Collaboration, 2002). However, aspirin is ineffective in approximately 20% of the patients, and clopidogrel should be prescribed.
- The treatment of unstable angina (preinfarction angina) requires clopidogrel in addition to aspirin, as well as the consideration of low-molecular weight (LMW) heparin and revascularisation.
- In primary prevention, aspirin can prevent myocardial infarctions but increases the risk of gastrointestinal bleeding and appears to increase the risk

of hemorrhagic stroke. The net benefit of aspirin increases with increasing cardiovascular risk (Hayden et al., 2002; Berger et al. 2006) [A].

### Choosing the Medication

- Sublingual or aerosol nitrates that are classically used for acute episodes should also be used for prophylaxis.
- A selective beta-blocker reduces both heart rate and blood pressure. It will also have a beneficial effect on tremor and migraine. Intermittent claudication is not a contraindication, unless the ischaemia is critical. The target heart rate is about 60 bpm at rest and below 120 bpm during exercise. With advancing age the dose can usually be reduced. Beta-blockers are also the first-line drugs for the treatment of arrhythmias of CHD patients. Heart failure is not a contraindication. Carvedilol may be the best choice in these cases. In heart failure an ACE inhibitor is usually combined with a beta-blocker. Beta-blockers are not only a symptomatic therapy, but they also reduce the risk of reinfarctions and sudden deaths in MI survivors by 10 to 30% (Freemantle et al., 1999). The prognosis is also improved in CHD patients who have not suffered an MI.
- Calcium-channel blockers may be considered if beta-blockers are unsuitable (Heidenreich et al., 1999) [B]. Of the older classic calcium-channel blockers, diltiazem is often chosen. Angina after a non-Q-wave infarction has been considered as its special indication. New dihydropyridine derivatives (amlodipine, felodipine, isradipine, nisoldipine) can be combined with beta-blockers in the treatment of stable angina, particularly if the patient is hypertensive. The effect of calcium antagonists on the prognosis is not as well documented as that of beta-blockers (Held & Yusuf, 1994) [B].
- A long-acting nitrate can be combined with a beta-blocker when the latter is not sufficient alone, or used instead of a beta-blocker when the drug is not tolerated. The nitrate is administered at the time when symptoms occur most often, which is often during the daytime. The usual dose is 20 to 40 (to 60) mg/day. A nitrate patch can be used to treat nocturnal angina. The patch should be removed in the morning to avoid the development of nitrate tolerance. For the same reason a pause should be kept in the administration of long-acting nitrates, for example, in the evening or at night. Nitrates are symptomatic therapy and are not needed if the patient is asymptomatic. They improve exercise tolerance but probably not the prognosis.
- The combination of beta-blockers, calcium-channel blockers, and long-acting nitrates (triple therapy) is usually more harmful than beneficial.

### Revascularisation

- Coronary angiography is warranted if the patient, whilst receiving appropriate medication, has troublesome ischaemic chest pain, and myocardial ischaemia has been verified (e.g., by exercise tolerance test). The method of revascularization is defined by coronary anatomy and the location and number of stenoses as confirmed by coronary angiography.
- Invasive treatments (coronary artery bypass grafting [CABG] and percutaneous transluminal coronary angioplasty [PTCA]) are only directed at the symptoms of coronary heart disease. Treatment according to the aetiology includes the management of all risk factors.

### Coronary Artery Bypass Grafting (CABG)

- Stenosis of the left main coronary artery (LCA) or three-vessel disease, which is of equal significance, are established indications for surgery (Yusuf et al., 1994; The Wessex Institute for Health Research and Development, 1998).
- CABG is often a better option (Sim, et al., 1995; Bakhai, et al., 2005) [C] if the patient has several total occlusions, the coronary anatomy is unfavourable for PTCA, or if the patient has diabetes, uraemia, significant left ventricular dysfunction, or a significant valvular disease (Baker et al., 1994).
- Minimally invasive off-pump bypass grafting (Mack, Osborne, & Shennib, 1998) [C] (Off-pump coronary artery bypass grafting [OP-CAB]) is a new surgical method that does not require the use of the heart-lung machine, and thoracotomy is not needed.
- A certain part of CABG patients will develop the so called postpericardiotomy syndrome (PPS) in a few weeks after the operation. The condition is easily managed with corticosteroids (see the Finnish Medical Society Duodecim guideline "Follow-Up of a Revascularized Patient").

### Percutaneous Transluminal Coronary Angioplasty (PTCA)

- 1 to 2 vessel CAD is an established indication for PTCA (The Wessex Institute for Health Research and Development, 1998).
- If the operative risks are high (e.g., difficult pulmonary disease, age), PTCA can be performed in LCA stenosis and in three-vessel disease.
- A post CABG patient with symptomatic coronary stenosis is primarily treated with PTCA.
- Insertion of a stent is an important part of PTCA. Over 80% of patients are fitted with stents. This has greatly diminished the number of complications and risk of restenosis. In most cases, a drug-eluting stent, which is impregnated with a smooth muscle growth inhibitor is used (Indolfi, Pavia, & Angelillo, 2005). [A] The use of drug-eluting stents has further extended the indications for PTCA.
- Acute MI: a large infarction that does not respond to thrombolysis (rescue PTCA) or the patient has a contraindication to thrombolysis and is at risk of an extensive anterior infarction.
  - Primary PTCA is increasingly replacing thrombolysis (Grines et al., 2003; Keeley, Boura, & Grines, 2003) [A], which is clearly not as efficient as PTCA in achieving patency of the vessels. Long-term outcomes also favour PTCA, largely due to stenting (The Wessex Institute for Health Research and Development, 1998; "Coronary artery stents, 1998) [B] (Grines et al., 1999; Robinson & Timmis, 2000).

### Related Evidence

- C-reactive protein may have independent value as a predictor of cardiovascular disease risk, but conclusive evidence on its role in risk assessment is lacking (ICSI, 2003; HTA-20030537, 2004; Blue Cross Blue Shield, 2003; HTA-20030742, 2004; Health Technology Advisory Committee, 2002; HTA-20030446, 2004) [C].

- Low glycaemic index diets may slightly reduce total cholesterol and HbA1c, but evidence is insufficient to recommend such diets for the purpose of improving risk factors for CHD (Kelly et al., 2004) [C].
- Dietary or supplemental omega 3 fats may not alter total mortality, combined cardiovascular events, or cancers in people with, or at high risk of, cardiovascular disease or in the general population (Hooper et al., 2004) [C].
- Self-reported dyspnea at cardiac stress testing appears to be an independent predictor of the risk of death from cardiac or any causes (Abidov, et al., 2005) [B].
- Dietary advice from health personnel appears to be effective in achieving modest dietary change and cardiovascular risk reduction (Brunner, et al., 2005; Brunner et al., 1997) [B].

#### Definitions:

#### Levels of Evidence

- Strong research-based evidence. Multiple relevant, high-quality scientific studies with homogenic results.
- Moderate research-based evidence. At least one relevant, high-quality study or multiple adequate studies.
- Limited research-based evidence. At least one adequate scientific study.
- No research-based evidence. Expert panel evaluation of other information.

#### CLINICAL ALGORITHM(S)

None provided

### EVIDENCE SUPPORTING THE RECOMMENDATIONS

#### REFERENCES SUPPORTING THE RECOMMENDATIONS

[References open in a new window](#)

#### TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

Concise summaries of scientific evidence attached to the individual guidelines are the unique feature of the Evidence-Based Medicine Guidelines. The evidence summaries allow the clinician to judge how well-founded the treatment recommendations are. The type of supporting evidence is identified and graded for select recommendations (see the "Major Recommendations" field).

### BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

#### POTENTIAL BENEFITS

- Appropriate diagnosis and treatment of angina pectoris and coronary artery disease
- Decreased morbidity and mortality associated with angina pectoris and coronary artery disease

- Cardiovascular risk reduction

## POTENTIAL HARMS

### Adverse Effects of Medications

- Beta-blockers and calcium antagonists can cause adverse effects.
- Aspirin increases the risk of gastrointestinal bleeding and appears to increase the risk of hemorrhagic stroke.

### Coronary Artery Bypass Grafting (CABG)

A certain part of CABG patients will develop the so called postpericardiotomy syndrome (PPS) in a few weeks after the operation. The condition is easily managed with corticosteroids.

### Exercise Tests

Exercise tests have many limitations and interpretation problems.

## IMPLEMENTATION OF THE GUIDELINE

### DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

## INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

### IOM CARE NEED

Getting Better  
Living with Illness  
Staying Healthy

### IOM DOMAIN

Effectiveness  
Patient-centeredness

## IDENTIFYING INFORMATION AND AVAILABILITY

### BIBLIOGRAPHIC SOURCE(S)

Finnish Medical Society Duodecim. Coronary heart disease (CHD): symptoms, diagnosis and treatment. In: EBM Guidelines. Evidence-Based Medicine [CD-ROM]. Helsinki, Finland: Duodecim Medical Publications Ltd.; 2006 Apr 28 [Various].

## ADAPTATION

Not applicable: The guideline was not adapted from another source.

## DATE RELEASED

2001 April 30 (revised 2006 Apr 28)

## GUIDELINE DEVELOPER(S)

Finnish Medical Society Duodecim - Professional Association

## SOURCE(S) OF FUNDING

Finnish Medical Society Duodecim

## GUIDELINE COMMITTEE

Editorial Team of EBM Guidelines

## COMPOSITION OF GROUP THAT AUTHORED THE GUIDELINE

Primary Author: Editors

## FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

Not stated

## GUIDELINE STATUS

This is the current release of the guideline.

This guideline updates a previous version: Finnish Medical Society Duodecim. Coronary heart disease (CHD): symptoms, diagnosis and treatment. In: EBM Guidelines. Evidence-Based Medicine [CD-ROM]. Helsinki, Finland: Duodecim Medical Publications Ltd.; 2006 Mar 14 [Various].

## GUIDELINE AVAILABILITY

This guideline is included in a CD-ROM titled "EBM Guidelines. Evidence-Based Medicine" available from Duodecim Medical Publications, Ltd, PO Box 713, 00101 Helsinki, Finland; e-mail: [info@ebm-guidelines.com](mailto:info@ebm-guidelines.com); Web site: [www.ebm-guidelines.com](http://www.ebm-guidelines.com).

## AVAILABILITY OF COMPANION DOCUMENTS

None available

## PATIENT RESOURCES

None available

## NGC STATUS

This summary was completed by ECRI on August 28, 2001. The information was verified by the guideline developer as of October 26, 2001. This summary was updated by ECRI on December 9, 2002, December 29, 2003, September 30, 2004, February 21, 2005, and July 13, 2006.

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